

An Heterogeneous SIS Model for Directed Networks and Optimal Curing Policy

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We investigate the influence of the contact network structure on the spread of epidemics over an heterogeneous population. In our model, the epidemic process spreads over a directed weighted graph. A continuous-time individual-based susceptible–infected–susceptible (SIS) is analyzed using a first-order mean-field approximation.

First, we consider a network with general topology in order to investigate the epidemic threshold and the stability properties of the system. Then, we analyze the case of a community network relying on the graph-theoretical notion of equitable partition. We show that, in this case, the epidemic threshold can be computed using a lower-dimensional dynamical system. Moreover we prove that the positive steady-state of the original system, that appears above the threshold, can be computed using this lower-dimensional system.

In the second part of the work, we leverage on our model to derive a cost-optimal curing policy, in order to prevent the disease from persisting indefinitely within the population. The solution of this optimization problem is obtained by formulating a convex minimization problem on a general but symmetric network structure. Finally, in the case of a two-level optimal curing problem an algorithm is designed with a polynomial time complexity in the network size.

Index Terms—heterogeneous SIS model, graph spectra, equitable partitions, convex optimization

I. INTRODUCTION

Several analytic studies in the literature have determined the conditions for the appearance of endemic infectious states over a population under the assumptions of homogeneous infection and recovery rates.

However, in many real situations, e.g., in social, biological and data communications networks, homogeneity is a demanding assumption and it appears more appropriate to consider instead an heterogeneous setting [1]. A concise overview on the literature considering heterogeneous populations can be found in [2], [3].

The objective of this work is to investigate the influence of the population contact network onto the development of epidemics and how it is possible to control it, by taking into account the topology of the network. A given directed weighted graph captures the interaction between individuals in a population and the large heterogeneity in the intensity of the connections.

We consider a continuous-time susceptible–infected–susceptible (SIS) epidemic process where

individual nodes interact according to prescribed heterogeneous and possibly asymmetric contact rates between them. The basic SIS model assumes that an individual can be repeatedly infected, recover and yet be infected again. This is the case, e.g., of several bacterial infections or computer viruses, i.e., infections for which, once cured, individuals cannot fend off subsequent attacks by the same agent. For computer networks, lack of anti-virus software leaves computers susceptible to the same malware infections [4].

The model is analyzed using a first-order mean-field approximation. First, we consider a network with general topology in order to investigate the epidemic threshold and the stability properties of the system.

In the second part of this work, we consider the case when a the contact network is shaped by an existing community network. The gross structure of hierarchical networks of this kind can be described by a quotient graph. Our investigations are based on the graph-theoretical notion of equitable partition. We show how possible symmetries in a directed weighted graph influence the system of differential

equations that solve for the evolution in time of the infection probability of each node. We use the word “symmetry” to refer to a certain structural regularity of the graph connectivity [5]. We use such regularity for reducing the original dynamical system to a lower dimensional one. We show that the epidemic threshold can be computed from this lower dimensional dynamical system, as well as the positive steady-state solution of the original system, that appears above the threshold.

Finally, with the aim of removing the epidemics from a population, we suppose that different curing rates can be chosen depending on the community network structure, and they can be optimized for a certain cost function. In the symmetric case, in particular, it is possible to use a convex optimization approach [6] to efficiently control the curing rate. For the case of two level curing rates, a scalable bisection algorithm is able to provide an ε -approximation in polynomial time in the input size.

A. Homogeneous SIS mean-field equation

In the homogeneous setting the epidemic spreads over a simple graph $G = (V, E)$ with edge set E and node set V . The order of G , denoted N , is the cardinality of V . Connectivity of graph G is conveniently expressed by the symmetric $N \times N$ adjacency matrix A .

The recovery process is a Poisson process with rate δ , and the infection process is a *per link* Poisson process where the infection rate between an healthy and an infected node is β . All the infection and recovery processes are independent, thus they compete for the production of an event (infection or recovery).

The state of the collective system of all nodes, i.e., the state of the network, is actually the joint state of all the nodes’ states. Since we assume that the infection and curing processes are of Poisson type, the SIS process, developing on a graph with N nodes, can be modeled as a continuous-time Markov process with 2^N states, covering all possible combinations in which N nodes can be infected [7], [8]. Unfortunately, the exponential growth with N of the state space poses severe limitations in order to determine the set of solutions for large, real networks [7], [8], [9].

For this reason a first order mean-field approximation (NIMFA) of the exact model is proposed

in [7]. For a special class of graphs it is possible to reduce the number of states (i.e., the number of equations) in the Markov chain and derive models that are either equivalent to the original system, but this is not generally the case (see [8]).

Basically, NIMFA replaces the actual infection rate for node i , $\beta \sum_{j=1}^N a_{ij} X_j(t)$ (where the sum is done on all the neighbor nodes), by its average rate $\beta \sum_{j=1}^N a_{ij} \mathbb{E}[X_j(t)]$, where $X_j(t)$ is the state variable representing the state of the node j [10].

Let $p_i(t)$ be the probability that a node i is infected at time t , $p_i(t) = \mathbb{E}[X_i(t)]$. According to the NIMFA model their dynamics obey to the following differential system

$$\frac{dp_i(t)}{dt} = \beta s_i(t)(1 - p_i(t)) - \delta_i(t), \quad i \in \{1, \dots, N\} \quad (1)$$

$$s_i(t) = \sum_{j=1}^N a_{ij} p_j(t), \quad (2)$$

where $s_i(t)$ represents the strength of the infection that may reach node i through the adjacency matrix $A = (a_{ij})$.

The time-derivative of the infection probability of node i consists of two competing processes:

- 1) while healthy (with probability $1 - p_i(t)$), all infected neighbors, whose average number is $s_i(t)$, try to infect node i at rate β ;
- 2) while node i is infected (with probability $p_i(t)$) it is cured at rate δ .

Epidemic threshold. For a network with finite order N the exact SIS Markov process will always converge towards its unique absorbing state, that is the zero-state where all nodes are healthy, so in the long run, the epidemics will be extinct. However the waiting time to absorption is a random variable whose distribution depends on the initial state of the system, on the parameters of the model and on the size of the population [11], [12]. In fact there is a critical value τ_c of the effective spreading rate $\tau = \beta/\delta$, whereby if τ is distinctly larger than τ_c , the time to absorption grows exponentially in N [11], while for τ er less than τ_c the epidemics dies out exponentially fast for sufficiently large time [13], [7], [14]. The exact computation of the exact quasi-stationary distribution is not analytically

tractable, as showed in [11]. Numerical simulations of SIS processes reveal that, even for fairly small networks ($N \simeq 100$) and when $\tau > \tau_c$, the overall healthy state is only reached after an unrealistically long time. Hence, the indication of the model is that, in the case of real networks, one should expect that above the epidemic threshold the extinction of epidemics is hardly ever attained [15], [16].

In the homogeneous setting NIMFA determines the epidemic threshold for the effective spreading rate as $\tau_c^{(1)} = \frac{1}{\lambda_1(A)}$, where the superscript (1) refers to the first-order mean-field approximation [7], [17]. When $\tau > \tau_c^{(1)}$, the mean-field equation (1) possess a second non-zero steady-state that reflects well the observed viral behavior [18] and that can be regarded as the analogous of the quasi-stationary distribution of the exact stochastic SIS model.

A consequence of replacing the random variable by its expectation in the mean-field approximations results in the neglect of correlations in the dynamic process: the mean-field approximation treats the joint probability $\mathbb{P}(X_i = 1, X_j = 1)$ as if X_i and X_j were independent, $\mathbb{P}(X_i = 1)\mathbb{P}(X_j = 1)$. As a consequence of this simplification, NIMFA yields an upper bound for the probability of infection of each node, as well as a lower bound for the epidemic threshold, i.e., $\tau_c \geq \tau_c^{(1)}$ [19]. If the states of the nearest nodes are sufficiently weakly dependent and the number of neighbors of node i , d_i (i.e., the degree of node i) is large enough so that the Lindeberg's Central Limit Theorem is applicable, then such replacement results in a good approximation. Informally, we can say that the mean-field approximation holds if in the underlying network, the degree of the nodes increase as the number of nodes N tends to infinity [20].

Moreover, evaluations on the variance of $\beta \sum_{i=1}^N a_{ij} X_j(t)$ in [7], shows that the deviations between the NIMFA model and the exact SIS are largest in some τ -region around the exact τ_c .

Further efforts have been made to satisfactorily quantify the accuracy of the first-order meanfield approximation [20]. Moreover, very recently, new approximate formula for the prevalence in the SIS model is given [21].

II. HETEROGENEOUS SIS ON NETWORKS

Now we include the possibility that the infection rate is link specific. Thus, we denote by β_{ij} the

infection rate of node j towards node i . We also exclude self-infection phenomena, thus we assume that $\beta_{ii} = 0$. Thus we allow for the epidemics to spread over a *directed weighted* graph. Moreover each node i recovers at rate δ_i , so that the curing rate is node specific.

As for the homogeneous SIS presented in Section I-A, the SIS model with heterogeneous infection and recovery rates is a Markovian process as well. The time for infected node j to infect any susceptible neighbor i is an exponential random variable with mean β_{ij}^{-1} . Also, the time for node j to recover is an exponential random variable with mean δ_j^{-1} .

In the same way as in the homogeneous setting, we provide the NIMFA approximation. In [1], a NIMFA model for the heterogeneous setting has been presented, where a node i can infect all neighbors with the same infection rate β_i . Here, instead, we include the possibility that the infection rates depend on the connection between two nodes, thus covering a much more general case.

The NIMFA governing equation for node i in the heterogeneous setting writes as

$$\frac{dp_i(t)}{dt} = \sum_{j=1}^N \beta_{ij} p_j(t) - \sum_{j=1}^N \beta_{ij} p_i(t) p_j(t) - \delta_i p_i(t), \quad i = 1, \dots, N \quad (3)$$

Let the vector $P = (p_1, \dots, p_N)^T$ and let $\bar{A} = (\bar{a}_{ij})$ be the matrix defined by $\bar{a}_{ij} = \beta_{ij}$ when $i \neq j$, and $\bar{a}_{ii} = -\delta_i$; moreover let $F(P)$ be a column vector whose i -th component is $-\sum_{j=1}^N \beta_{ij} p_i(t) p_j(t)$. Then we can rewrite (4) in the following form:

$$\frac{dP(t)}{dt} = \bar{A}P(t) + F(P). \quad (4)$$

Let

$$r(\bar{A}) = \max_{1 \leq j \leq N} \text{Re}(\lambda_j(\bar{A}))$$

be the *stability modulus* [22] of \bar{A} , where $\text{Re}(\lambda_j(\bar{A}))$ denotes the real part of the eigenvalues of \bar{A} , $j = 1, \dots, N$. We report a result from [22] that lead us to extend the stability analysis of NIMFA in [23] to the heterogeneous case.

Theorem 1. *If $r(\bar{A}) \leq 0$ then $P = 0$ is a globally asymptotically stable equilibrium point in $I_N = [0, 1]^N$ for the system (4). On the other hand if $r(\bar{A}) > 0$ then there exists a constant solution $P^\infty \in I_N - \{0\}$, such that P^∞ is globally asymptotically stable in $I_N - \{0\}$ for (4).*

III. COMMUNITY NETWORKS

A community structure is an important non-trivial topological feature of a complex networks. Indeed community structures are a typical feature of social networks, tightly connected groups of nodes in the World Wide Web usually correspond to pages on common topics, communities in cellular and genetic networks are related to functional modules [24].

Thus, in order to investigate this topological feature, we consider, in this section, that the entire population is partitioned into communities (also called households, clusters, subgraphs, or patches).

There is an extensive literature on the effect of network community structures on epidemics. A specific community structure may arise due to, for example, geographic separation. Models utilizing this structure are commonly known as “metapopulation” models (see, e.g., [25], [26], [27]). Such models assume that each community shares a common environment or is defined by a specific relationship. Some of the most common works on metapopulation regard a population divided into households with two level of mixing ([28], [29], [30]). This model typically assumes that contacts, and consequently infections, between nodes in the same group occur at a higher rate than those between nodes in different groups [31]. Thus, groups can be defined, e.g., in terms of spatial proximity, considering that between-group contact rates (and consequently the infection rates) depend in some way on spatial distance. In this type of heterogeneous contact networks each node can be theoretically infected by any other node. However, an underlying network contact structure may provide a more realistic approach for the study of the evolution of the epidemics, where infection can only be transmitted by node directly linked by an edge [31]. In turn, an important challenge is how to consider a realistic underlying structure and appropriately incorporate the influences of the network topology on the dynamics of epidemics [32], [33], [34], [35], [36], [37].

To this aim, we analyzed in [23], [38] the dynamics of epidemics on networks that are partitioned into local communities, through the first-order mean-field approximation discussed in Section I-A. Our investigation has been based on the graph-theoretical notion of *equitable partition* [39], [40], [5].

For an undirected graph, let $\pi = \{V_1, \dots, V_n\}$ be a partition of the node set V , which is assumed to be given a priori; π is called equitable if the subgraph G_i of $G(V, E)$ induced by V_i is regular for all i 's. Furthermore, for any two subgraphs G_i and G_j , whenever there exists at least one connection between nodes in the first and second subgraph, then each node in G_i is connected with the same number of nodes in G_j .

In [23], [38] two-level infection rates have been considered: an intra-community infection rate and an inter-community infection rate. In the network structures hereafter, we generalize the model to more than two levels of mixing.

Such network structures can describe models consisting of multiple smaller sub-populations such as, e.g., households, workplaces, or classes in a school, when the internal structure of each community is represented by a complete graph (members of a small community usually know each other) and all the nodes of adjacent communities are mutually linked (all member of adjacent communities may potentially come into contact). Equitable partitions can be observed also in the architecture of some computer networks where clusters of clients connect to single routers, whereas the router network has a connectivity structure with the nodeal degree constrained by the number of ports.

In the following section we define the equitable partition for the case of a directed weighted networks, extending the analysis in [23] to this framework.

A. Equitable partitions for weighted directed networks

Now we extend the definition of equitable partitions to weighted directed graphs, starting from [5, Def. 8.24], where the definition is given for oriented weighted graphs [5, Def. A.1].

Let $\rho : E \rightarrow [0, \infty)$ be a given function that assigns to each arc $e = (v, w) \in E$ a *weight* $\rho(e) = \beta_{vw}$, then we have the following definition.

Definition 1. Let $G = (V, E)$ be a weighted directed graph. The partition $\pi = \{V_1, \dots, V_n\}$ of the node set V is called *inward equitable* or *outward equitable*

if for all $i, j \in \{1, \dots, n\}$, there are

$$c_{ij}^{in} \in \mathbb{R} \quad \text{s.t.} \quad \sum_{w \in V_j} \rho((v, w)) = c_{ij}^{in},$$

for all $v \in V_i$,

or

$$c_{ij}^{out} \in \mathbb{R} \quad \text{s.t.} \quad \sum_{w \in V_j} \rho((w, v)) = c_{ij}^{out},$$

for all $v \in V_i$,

respectively. The partition is called equitable if it is both inward and outward equitable, hence for all $i, j \in \{1, \dots, n\}$, there are

$$c_{ij} \in \mathbb{R} \quad \text{s.t.} \quad \sum_{w \in V_j} \rho((v, w)) + \rho((w, v)) = c_{ij},$$

for all $v \in V_i$.

We shall identify the set of all nodes in V_i with the i -th community of the whole population.

Remark 1. Let k_i be the order of V_i , $i = 1, \dots, n$. If the partition of the node set of a weighted di-graph is equitable, then for all $i, j \in \{1, \dots, n\}$,

$$k_i c_{ij}^{out} = k_j c_{ji}^{in}, \quad (5)$$

An equitable partition generates the *quotient graph* G/π , which is a *multigraph*, directed and weighted, with cells as vertices. There exists a quotient matrix related to the quotient graph that contains the relevant structural information of the networks.

Let us consider the $n \times n$ matrix $S = (s_{iv})$, where

$$s_{iv} = \begin{cases} \frac{1}{\sqrt{|V_i|}} & v \in V_i \\ 0 & \text{otherwise.} \end{cases}$$

where it follows that $SS^T = I$.

Now let us consider the transpose of the adjacency matrix of the weighted directed graph G , that is

$$A^T = \bar{A} + D, \quad (6)$$

where $D = \text{diag}(\delta_i)$, $i = 1, \dots, n$ is the curing rate matrix. Then the transpose of the *quotient matrix* of G (with respect to the given partition) is

$$Q^T := SAS^T.$$

We can write the following explicit expression for Q^T :

$$Q^T = \text{diag}(c_{ii}^{out}) + \left(\frac{k_i c_{ij}^{out}}{\sqrt{k_i k_j}} \right)_{i,j=1,\dots,n}. \quad (7)$$

By (5) we can write the matrix Q^T as

$$Q^T = \text{diag}(c_{ii}^{out}) + \left(\sqrt{c_{ij}^{out} c_{ji}^{in}} b_{ij} \right)_{i,j=1,\dots,n}. \quad (8)$$

Remark 2. We observe that matrix Q in (8) might not be symmetric, whereas in the case of undirected graphs it is always symmetric (see e.g. [40], [23]). Even though we have represented the most general definition of an equitable partition of the node set simpler situations can be represented. E.g., nodes of the same community may infect all nodes in another community with the same rate.

For the purpose of modeling, nodes of the quotient graph can represent communities, e.g., villages, cities or countries. Link weights in the quotient graph in turn provide the strength of the contacts between such communities. In particular, the weight of a link may be (a non-negative) function of the number of people traveling per day between two countries; in fact, the frequency of contacts between them correlates with the propensity of a disease to spread between nodes.

B. Dimensionality reduction of the dynamical system

When considering a population partitioned into communities, it may be appropriate to take into account the case where all nodes of a tagged community j have the same recovery rate δ_j , $j = 1, \dots, n$. In turn, such rate may differ from one community to the other. We remember that N is the total number of nodes in the network, whereas n is the number of communities.

Definition 2. Let us introduce the $1 \times n$ vector of nonzero curing rates $\bar{\Delta} = (\bar{\delta}_1, \dots, \bar{\delta}_n)$, that we shall call the *reduced curing rate vector*.

It is straightforward to adapt the proof in [23, Thm. 4.1] for the case of directed graphs. Following that result, when at time $t = 0$ the infection probability is equal for all nodes in the same community (and may differ from one community to the other), the number of equations in (4) can be reduced

by using the transpose of the quotient matrix Q^T . Hence, the reduced dynamical system writes

$$\begin{aligned} \frac{d\bar{p}_j(t)}{dt} &= (1 - \bar{p}_j(t)) \sum_{\substack{m=1 \\ m \neq j}}^n c_{jm}^{\text{out}} \bar{p}_m(t) + c_{jj}^{\text{out}} (1 - \bar{p}_j(t)) \bar{p}_j(t) \\ &\quad - \bar{\delta}_j \bar{p}_j(t), \quad j = 1, \dots, n \end{aligned} \quad (9)$$

where $\bar{p}_j(t)$ is the infection probability of a node of community j .

We know that

$$q_{jm}^T = \frac{k_j c_{jm}^{\text{out}} b_{jm}}{\sqrt{k_j k_m}}$$

hence

$$c_{jm}^{\text{out}} b_{jm} = \frac{\sqrt{k_j k_m}}{k_j} q_{jm}^T = \left(\frac{k_m}{k_j} \right)^{1/2} q_{jm}^T$$

Thus we can write (9) in the following matrix form

$$\frac{d\bar{P}(t)}{dt} = (\tilde{Q} - \bar{D}) \bar{P}(t) - \text{diag}(\bar{P}(t)) \tilde{Q} \bar{P}(t), \quad (10)$$

where $\tilde{Q} = \text{diag} \left(\frac{1}{\sqrt{k_j}} \right) Q^T \text{diag}(\sqrt{k_j})$. It is immediate that $\sigma(Q^T) = \sigma(\tilde{Q})$ (where with $\sigma(A)$ we indicate the spectrum of the matrix A).

Now let us define the $1 \times N$ curing rates vector $\Delta = (\delta_1, \dots, \delta_N)$, where $\delta_z = \bar{\delta}_j$ for all $z \in V_j$ and $j = 1, \dots, n$, and $D = \text{diag}(\Delta)$ is the $N \times N$ curing rate matrix. Then, the following holds:

Lemma 1. *Let $\pi = \{V_1, \dots, V_n\}$ be an equitable partition. Let A^T and Q^T be weighted matrices as in (6) and (8) respectively. Then it holds*

- i) $(A^T - D)S^T = S^T(Q^T - \bar{D})$.
- ii) For all $\lambda \in \mathbb{C}$ and all $x \in \mathbb{C}^n$

$$(Q^T - \bar{D})x = \lambda x \quad \text{if and only if} \quad (A^T - D)S^T x = \lambda S^T x.$$

Proof. i) We first prove that $AS^T = S^TQ$. In fact, if $i \in V_h$, it holds

$$(A^T S^T)_{i,j} = \frac{c_{hj}^{\text{out}}}{\sqrt{k_j}} \quad (11)$$

$$(S^T Q^T)_{i,j} = \frac{1}{\sqrt{k_h}} q_{hj}^T = \frac{c_{hj}^{\text{out}}}{\sqrt{k_j}} \quad (12)$$

We further note that $(DS^T)_{ih} = (S^T \bar{D})_{ih} = \frac{1}{\sqrt{k_h}} \delta_h$, if $i \in V_h$, otherwise $(DS^T)_{ih} = 0$. Thus the statement holds.

ii) By using the result in i), the proof in [40, Thm. 2.2] applies. \square

Now let us consider the system of N differential equations (4). We can prove that, in the case of a graph whose node set has an equitable partition, and regardless of initial conditions, it is possible to determine the critical threshold for (4), applying Thm. 1, directly on the reduced system (10).

Proposition 1. *The elements of the curing rates vector $\Delta = (\delta_1, \dots, \delta_N)$, that determines the critical threshold of (4), are identified by the elements of $\bar{\Delta} = (\bar{\delta}_1, \dots, \bar{\delta}_n)$, in such a way that $\delta_z = \bar{\delta}_j$ for all $z \in V_j$, $j = 1, \dots, n$, for which*

$$r(Q^T - \bar{D}) = 0, \quad (13)$$

where r is the stability modulus.

Proof. Basically, by Theorem 1, we have to show that

$$r(A^T - D) = r(\tilde{Q} - \bar{D}) = r(Q^T - \bar{D}). \quad (14)$$

We first prove that

$$r(Q^T - \bar{D}) = r(A^T - D). \quad (15)$$

By the definition of Q we have that

$$S(A^T - D)S^T = SA^T S^T - SDS^T = Q^T - \bar{D}.$$

Now, let $c \in \mathbb{R}$ such that both $a_{zz}^T - \delta_z + c \geq 0$, for all $z = 1, \dots, N$ and $q_{ii}^T - \bar{\delta}_i + c \geq 0$ for all $i = 1, \dots, n$. Let us define $A^T - D^T + cI_{N \times N} = \hat{A}^T$ and $Q^T - \bar{D} + cI_{n \times n} = \hat{Q}^T$. \hat{A}^T and \hat{Q}^T are non negative and irreducible matrices (see i) in Proposition 2). We order the eigenvalues of \hat{Q}^T so that $|\lambda_1(\hat{Q}^T)| \geq |\lambda_2(\hat{Q}^T)| \geq \dots \geq |\lambda_n(\hat{Q}^T)|$, and similarly for \hat{A}^T . By the Perron-Frobenius theorem, the eigenvalue of maximum modulus of an irreducible and non negative matrix is real and positive and its corresponding eigenvector, the Perron vector, is the unique (up to a factor) strictly positive eigenvector of the matrix. Hence there exists an eigenvector $\omega > 0$ of \hat{Q}^T corresponding to $\lambda_1(\hat{Q}^T)$, i.e. $w_i > 0$, for all $i = 1, \dots, n$.

By ii) in Lemma 1 and since, $S^T I_{n \times n} = I_{N \times N} S^T$, we have that $S^T \omega > 0$ is the eigenvector of \hat{A}^T corresponding to $\lambda_1(\hat{Q}^T)$. However, since $S^T \omega$ is strictly positive, it must be the Perron vector of \hat{A}^T , consequently $\lambda_1(\hat{A}^T) = \lambda_1(\hat{Q}^T)$.

It can be immediately shown that

$$r(\hat{Q}^T) = \lambda_1(\hat{Q}^T) = \lambda_1(\hat{A}^T) = r(\hat{A}^T),$$

and that

$$r(Q^T - \overline{D}) + c = r(\hat{Q}^T) = r(\hat{A}^T) = r(A^T - D) + c, \quad (16)$$

thus (15) holds.

Now we prove that

$$r(\tilde{Q} - \overline{D}) = r(Q^T - \overline{D}). \quad (17)$$

Let the matrix $\Lambda^{\frac{1}{2}} = \text{diag}(k_i)$. For any n -dimensional vector v and scalar $\lambda \in \mathbb{C}$ we have that

$$\begin{aligned} (\tilde{Q} - \overline{D})v = \lambda v &\Leftrightarrow (\Lambda^{-\frac{1}{2}}Q\Lambda^{\frac{1}{2}} - \overline{D})v = \lambda v \Leftrightarrow \\ (Q\Lambda^{\frac{1}{2}} - \overline{D}\Lambda^{\frac{1}{2}})v &= \lambda\Lambda^{\frac{1}{2}}v \Leftrightarrow \\ (Q - \overline{D})(\Lambda^{\frac{1}{2}}v) &= \lambda(\Lambda^{\frac{1}{2}}v), \end{aligned}$$

hence $\lambda \in \sigma(\tilde{Q} - \overline{D}) \iff \lambda \in \sigma(Q - \overline{D})$, so that (17) is verified.

In conclusion, from (17) and (15) it follows (14).

□

We underline that if A is an $N \times N$ irreducible and non negative matrix, and D a diagonal matrix with positive entries, then the eigenvalue $\lambda \in \sigma(A - D)$, such that $\text{Re}(\lambda) = r(A - D)$, is real. This fact also follows from (16).

Since the quotient matrix and the adjacency matrix have the same stability modulus (and so their transposed do), a computational advantage is obtained in the calculation of the critical threshold of the system (4).

Furthermore, by [23, Corollary 4.2], irrespective of the initial conditions of nodes in the same community, it is sufficient to compute the positive steady-state vector \overline{P}_∞ of the reduced system (10) to obtain that of the original system (4). Indeed, as time elapses all nodes in the same community tend to have the same infection probabilities, thus the components of the steady-state vector P_∞ corresponding to nodes in the same community are equal.

These results on equitable partitions can also be useful to adopt an efficient curing policy that takes into account the community structure of the population. To this aim, in the next section, we consider the important issue of controlling an epidemic by taking into account the population contact network.

IV. OPTIMAL CURING POLICIES

An important challenge in epidemiology is to understand how to control epidemic outbreaks, both in public health and in other domains, such as, e.g., protection of computer networks from electronic viruses. In particular, since the network structure plays a crucial role in the diffusion of epidemics, not always a uniform distribution of resources among nodes will work efficiently in order to eradicate the infection, or reduce the number of infected nodes [41], [42].

Furthermore, curing costs may vary from node to node. Thus, our objective is to determine a cost-optimal distribution of resources yet able to prevent the disease from persisting indefinitely in the population.

Borgs et al. [42] characterize the optimal distribution of a fixed amount of antidote in a given contact network by a probabilistic analysis, based on the theory of contact processes. Wan et al. [43] consider the problem of allocating limited control resources in a networks, considering the topological structure, so as to maximize the speed at which the virus is eliminated. They design optimal strategies to control the diffusion of the virus using eigenvalue sensitivity analysis ideas and a constrained optimization method. Gourdin et al. [44] and Sahnen et al. [45] the N -intertwined model is used to analyze and control the spread of an SIS epidemic model.

Drakopoulos [46], [47] the authors consider the propagation of an epidemic process on a network and study the problem of dynamically allocating a fixed curing budget to the nodes of the graph, at each time instant, towards the objective of minimizing the expected extinction time of the epidemic. In the case of bounded degree graphs, they provide a lower bound on the expected time to extinction under any such dynamic allocation policy.

In our work, we present an optimal resource allocation strategy, taking into account the results of Theorem 1. First, we define a cost function which measures the cost that it is necessary to cover in order to distribute resources to all nodes. Let $f_i(\delta_i)$ be a convex and monotonically increasing function with respect to δ_i , whose value represents the effort of modifying the recovery rate of a node i ; thus to allocate curing resources at a given node increases its recovery rate.

This model fits the case of disease treatment

plans: policy makers can distribute different amount of resources (e.g. money for medicines, medical and nursery staff, etc,...), in a network of hospitals, or they can design a different health program for different districts, cities, or nations in the case of a timely mass prophylaxis plan. For instance, in the US, pharmaceutical supply caches and production arrangements have been pre-designated, in order to be used for large-scale ongoing prophylaxis and/or vaccination campaigns in case of sudden intentional or natural outbreaks disease [48].

The cost functional is the cumulative sum over the nodes' set

$$U(\Delta) = \sum_{i=1}^N c_i f_i(\delta_i) \quad (18)$$

where Δ is the curing rate vector, whereas $c_i > 0$, for $i = 1, \dots, N$, is the cost for curing node i

Hereafter, we will assume that $\beta_{ij} = \beta_{ji}$, for all $i, j = 1, \dots, N$, i.e., the weighted adjacency matrix A is symmetric, and consequently all its eigenvalues are real.

By Theorem 1 we know that if $\lambda_1(A - \text{diag}(\Delta)) \leq 0$, then the epidemics will go extinct. As we explain in Section I-A the critical threshold for the mean-field model is a lower bound of the threshold of the exact Markov model. Thus the condition $\lambda_1(A - \text{diag}(\Delta)) \leq 0$ corresponds, in the exact stochastic model, to a region where the infectious process dies out exponentially fast for sufficiently large times. We recall that, instead, above the exact threshold the overall-healthy state is reached only after an unrealistically long time. Hence, in order to find a cost-optimal distribution of resources that guarantees the extinction we seek for the solution of the following problem.

Problem 1 (Eigenvalue Constraint Formulation). *Find $\Delta \geq 0$ which solves*

$$\begin{aligned} & \text{minimize} && U(\Delta) \\ & \text{subject to:} && \lambda_1(A - \text{diag}(\Delta)) \leq 0, \quad \Delta \geq 0 \end{aligned}$$

Problem 1 can be reformulated as a semidefinite program, that is a convex optimization problem [49]. In fact $\Delta = \sum_{i=1}^N \Delta_i \text{diag}(\mathbf{e}_i)$, where Δ_i is the i -th component of Δ and \mathbf{e}_i is the i -th element of the standard basis so that $\text{diag}(\mathbf{e}_i) \geq 0$. Hereafter, as in [50], the inequality sign in $M \geq 0$, when M is a matrix, means that M is positive semidefinite.

Thus we can express the optimization problem with eigenvalue constraint as a semidefinite programming problem.

Problem 2 (Semidefinite Programming Formulation). *Find Δ which solves*

$$\begin{aligned} & \text{minimize} && U(\Delta) \\ & \text{subject to:} && \text{diag}(\Delta) - A \geq 0 \\ & && \Delta \geq 0 \end{aligned}$$

The feasibility of the problem is always guaranteed, as showed in the following

Theorem 2 (Feasibility). *Problem 2 is feasible.*

Proof. We define $l_{\max} := \max_i \sum_j a_{ij}$ and choose $\Delta = l_{\max} \mathbf{u}$, where \mathbf{u} is the all-one vector: $D = l_{\max} I_N$. Then for any vector $w = \sum_{i=1}^N z_i v_i$ where $\{v_1, \dots, v_N\}$ is an eigenvector basis of A , it holds

$$\begin{aligned} w^T(A - D)w &= w^T \left(\sum \lambda_i(A) z_i v_i - l_{\max} w \right) \\ &\leq (\lambda_1(A) - l_{\max}) \|w\|^2 \leq 0, \end{aligned}$$

where the last inequality follows since $\lambda_1(A) \leq \max_i \sum_j a_{ij}$. Hence the chosen vector satisfies the constraint and we can assert that the feasible region is not empty. \square

Since the problem is feasible there is always an optimal point on the boundary [49] and, by the fundamental result of convex optimization, any locally optimal point of a convex problem is globally optimal [50, Sec. 4.4.2].

A semidefinite programming (SDP) approach is adopted also by Preciado et al. for the study of optimal network immunization in [6], where they provide an optimal vaccine allocation in an arbitrary networks. In particular they consider that each node i can infect all its neighbors with the same infection rate β_i , and describe the minimization of an arbitrary convex function of the infection rates. In [51], they generalized the convex formulation via Geometric Programming techniques to weighted, directed, strongly (and not necessarily strongly) connected networks to compute the cost and speed optimal allocation of vaccines and/or antidotes. Enyioha et al. [52] propose a distributed solution to the vaccine and antidote allocation problem to contain an epidemic outbreak in the absence of a central social planner. Each node locally computes its optimum investment in vaccine and antidotes needed

to globally contain the spread of an outbreak, via local exchange of information with its neighbors.

The next section extends the optimization approach to a network partitioned into communities. Indeed, in most practical situations, it appears reasonable to consider curing policies which apply *per community* (i.e., per hospital, school, village, or city, etc,...), rather than curing policies which apply per node.

A. Optimization for Networks with Equitable Partitions

In this section we consider a heterogeneous curing control per community.

Let us consider $\beta_{V_i V_j}$ as the rate at which nodes belonging to community j infect those belonging to community i , and let us assume that $\beta_{V_i V_j} = \beta_{V_j V_i}$, $i, j = 1, \dots, n$. Hence, the quotient matrix Q is symmetric and has real eigenvalues.

Let us consider the $1 \times n$ reduced curing rate vector $\bar{\Delta}$ and the $1 \times n$ cost vector c , where component c_j is the curing cost per node in the j -th community. The cost function writes

$$U(\bar{\Delta}) = \sum_{j=1}^n c_j k_j f(\delta_j)$$

Thus, $U(\bar{\Delta})$ is the cost for curing all elements of each community j at rate δ_j , $j = 1, \dots, n$. We seek for the solution of the following

Problem 3 (Eigenvalue Constraint Formulation). Find $\bar{\Delta} \geq 0$ which solves

$$\begin{aligned} & \text{minimize} && U(\bar{\Delta}) \\ & \text{subject to:} && \lambda_1(Q - \text{diag}(\bar{\Delta})) \leq 0, \quad \bar{\Delta} \geq 0 \end{aligned}$$

which also writes

Problem 4 (Semidefinite Programming Formulation). Find $\bar{\Delta} \geq 0$ which solves

$$\begin{aligned} & \text{minimize} && U(\bar{\Delta}) \\ & \text{subject to:} && \text{diag}(\bar{\Delta}) - Q \geq 0 \\ & && \bar{\Delta} \geq 0 \end{aligned}$$

Thm. 2 guarantees the feasibility of the problem. Semidefinite programs can be solved using standard tools [53]. For systems of moderate size (e.g., on the order of $n = 100$ communities), the number of involved variables does not represent a serious

performance bottleneck and standard solvers perform well in practice. Scalability properties limit the usage of semidefinite programming for very large graphs, which in general need not to be sparse.

B. Two-level curing problem

We consider a simpler version of Problem 4, where we assume that there exist two types of communities. Communities of the first type are subject to curing rate δ_0 whereas communities of the second type are subject to curing rate δ_1 . For convenience we define *central* communities, those whose elements have curing rate δ_0 , and *terminal* communities those whose elements have curing rate δ_1 .

This kind of situation can be well represented by a network that is, e.g., bipartite (where each node, e.g., represent a full-meshed community), or an interconnected stars network, i.e. interconnecting star graphs, where stars' central nodes may be connected among themselves (see Fig 1).

Let us note that the Barabási-Albert graph model [54], that captures the power-law degree distribution often seen (or approximately seen) in real-world networks, can be seen as a set of hubs with star graph features [55].

Bipartite networks, instead, can be used to understand the spreading of sexually transmitted diseases, in which the population is naturally divided in males and females and the disease can only be transmitted between nodes of different kinds. Bipartite networks can also represent the spreading of diseases in hospitals, in which one type of node accounts for (isolated) patients and the other type for caregivers, or some vector-borne diseases, such as malaria, in which the transmission can only take place between the vectors and the hosts [56].

We consider the following partition of the node set: set $\pi_0 = \{V_1^0, \dots, V_m^0\}$, and set $\pi_1 = \{V_1, \dots, V_{m'}\}$. We assume that the node set partition $\pi = \pi_0 \cup \pi_1$ is equitable.

Thus, let us consider the curing matrix $D = \text{diag}(\delta_0 \mathbf{1}_m, \delta_1 \mathbf{1}_{m'})$. We also define

$$I_m^0 = \begin{bmatrix} I_m & 0 \\ 0 & 0 \end{bmatrix}, \quad I_{m'}^1 = \begin{bmatrix} 0 & 0 \\ 0 & I_{m'} \end{bmatrix}$$

where I_m is the identity matrix of order m . We can write the semidefinite programming for the two-level curing rates, shortly the $2D$ curing problem, as follows:

Problem 5 (Semidefinite Programming 2D Formulation). Find $\Delta_2 = (\delta_0, \delta_1)$ which solves

$$\begin{aligned} & \text{minimize} && U(\Delta_2) \\ & \text{subject to:} && \delta_0 I_m^0 + \delta_1 I_{m'}^1 - Q \geq 0 \\ & && \Delta_2 \geq 0 \end{aligned}$$

where the cost function is

$$U(\Delta_2) = \sum_{V_j \in \pi_0} c_0 k_j f_0(\delta_0) + \sum_{V_z \in \pi_1} c_1 k_z f_1(\delta_1),$$

and c_0 is the cost for curing a node belonging to $V_j \in \pi_0$ while c_1 is the cost for curing a node belonging to $V_z \in \pi_1$; $f_0(\delta_0)$ and $f_1(\delta_1)$ represent the effort to modify the recovery rate for nodes in central communities and nodes in terminal communities, respectively.

C. Examples

1. A simple example of the optimal solution for the case of a community network is that of a star graph, where we have two communities, one formed by the central node and the other by the leaf nodes.

Assuming that the infection rate is β , we have to find the value of δ_0 and δ_1 for which $\beta Q - D$ has the maximal eigenvalue which is equal to zero. The characteristic polynomial of $\beta Q - D$ for a star graph with k leaves is

$$p_\lambda(\beta Q - D) = \lambda^2 + (\delta_0 + \delta_1)\lambda + \delta_0\delta_1 - \beta^2 k$$

We observe that $\lambda = 0$ belongs to the spectrum of $\beta Q - D$ if and only if $\delta_0 = \beta^2 k / \delta_1$. This also ensures that the second eigenvalue is negative and, consequently, $\lambda = 0$ must be the largest eigenvalue of $\beta Q - D$. Now let us consider $f_0(\delta_0) = \delta_0$ and $f_1(\delta_1) = \delta_1$, then replacing the value of δ_0 obtained above in

$$U(\delta_0, \delta_1) = c_0 \delta_0 + c_1 k \delta_1,$$

and setting to zero the following derivative

$$U'(\delta_1) = -\frac{c_0 \beta^2 k^3}{\delta_1^2} + c_1 k,$$

we have that the linear cost optimization is solved for

$$\delta_0 = \beta k \sqrt{\frac{c_1}{c_0}}, \quad \delta_1 = \beta \sqrt{\frac{c_0}{c_1}}, \quad (19)$$

which in turn provides the optimal cost

$$U^* = \beta k \left(c_0 \sqrt{\frac{c_1}{c_0}} + c_1 \sqrt{\frac{c_0}{c_1}} \right) = 2\beta k (\sqrt{c_1 c_0})$$

We observe that the optimal cost is linear in the terminal community size k .

In the case of an uniform curing policy, where all nodes are cured at rate δ , we have that the value of δ such that $\lambda = 0$ is the largest eigenvalue of $\beta Q - D$ is equal to $\beta \sqrt{k}$, thus the value U_u of the total cost is

$$U_u = \beta \sqrt{k} (c_0 + c_1 k)$$

It is easy to see that the ratio U_u / U^* is increasing in $(1, \infty)$, moreover we can observe that

$$\frac{U_u}{U^*} = O(\sqrt{k}). \quad (20)$$

Hence it is clear that we have an advantage in considering a two-level curing policy, with respect to the uniform curing policy.

2. Now we consider an interconnected star network with two linked central nodes, where each terminal community has the same number of elements k . We set β as the infection rate between the central nodes and $\varepsilon \beta$ the infection rate between a central node and a node in its adjacent terminal community, where $\varepsilon > 0$.

After computing the characteristic polynomial of $\beta Q - D$ we can see that the zero eigenvalue belongs to the spectrum of $\beta Q - D$ provided that

$$\delta_0^2 \delta_1^2 - 2\beta^2 \delta_0 \delta_1 \varepsilon^2 k + \varepsilon^4 \beta^4 k^2 - \beta^2 \delta_1^2 = 0.$$

The values of δ_0 for which $\lambda = 0$ corresponds to the largest eigenvalue of $\beta Q - D$ is equal to $\delta_0 = \frac{\beta^2 \varepsilon^2 k}{\delta_1} + \beta$ and the linear cost optimization is solved for

$$\delta_0 = \beta \left(\varepsilon k \sqrt{\frac{c_1}{c_0}} + 1 \right), \quad \delta_1 = \varepsilon \beta \sqrt{\frac{c_0}{c_1}}$$

Consequently the optimal cost is

$$\begin{aligned} U^* &= 2\beta c_0 \left(\varepsilon k \sqrt{\frac{c_1}{c_0}} + 1 \right) + 2c_1 k \sqrt{\frac{c_0}{c_1}} \varepsilon \beta \quad (21) \\ &= 2\beta \sqrt{c_0 c_1} (\varepsilon (k + 1) + c_0) \end{aligned}$$

In the case of an uniform curing policy we have that the value of δ such that $\lambda = 0$ is the largest eigenvalue is $(\beta + \sqrt{\beta^2 + 4\beta^2 \varepsilon^2 k})/2$ and the value of the total cost is

$$U_u = c_0 \left(\beta + \sqrt{\beta^2 + 4\beta^2 \varepsilon^2 k} \right) + c_1 k \left(\beta + \sqrt{\beta^2 + 4\beta^2 \varepsilon^2 k} \right)$$

The ratio U_u/U^* is increasing in $(0, \infty)$, and again we have that

$$\frac{U_u}{U^*} = O(\sqrt{k})$$

3. Now we consider a bipartite graph. Basically, we have two communities and we denote by k_0 the number of elements in the community whose nodes have recovery rate δ_0 and k_1 the number of elements in the community whose nodes has recovery rate δ_1 . The optimal curing rates are

$$\delta_0 = \beta k_1 \sqrt{\frac{c_1}{c_0}}, \quad \delta_1 = \beta k_0 \sqrt{\frac{c_0}{c_1}},$$

and the optimal cost is

$$U^* = c_0 k_0 \beta k_1 \sqrt{\frac{c_1}{c_0}} + c_1 k_1 \beta k_0 \sqrt{\frac{c_0}{c_1}}.$$

In the case of an uniform curing policy the value of δ such that $\lambda = 0$ is the largest eigenvalue is

$$\delta = \beta \sqrt{k_0 k_1},$$

and the cost is

$$U_u = c_0 k_0 \beta \sqrt{k_0 k_1} + c_1 k_1 \beta \sqrt{k_0 k_1}.$$

In this case the asymptotic behavior of U_u/U^* for high values of k_0 and k_1 depends on the direction in which we move, thus we can not say anything in this regard.

D. Properties of the 2D curing problem

In the design of our algorithmic solution, we have leveraged on some basic properties of the 2D curing problem.

Lemma 2 (Monotonicity). *Let $\phi : \delta_0 \mapsto \phi(\delta_0)$ be the function that associates to each $\delta_0 \in \mathbb{R}^+$ the value $\delta_1 = \phi(\delta_0) \in \mathbb{R}^+$ such that $\lambda_1(Q - \text{diag}(\delta_0 \mathbf{1}_m, \delta_1 \mathbf{1}_{m'})) = 0$. Then ϕ is decreasing.*

Proof. Let $z > 0$ and assume that $\phi(\delta_0 + z) = \phi(\delta_0) + \zeta > \phi(\delta_0)$, for some $\zeta > 0$, i.e., that ϕ is not decreasing. From the definition of ϕ there exists $0 \neq w \in \ker(\text{diag}(((\delta_0 + z) \mathbf{1}_m, \phi(\delta_0 + z) \mathbf{1}_{m'}) - Q))$. Hence, we can write

$$\begin{aligned} w^T (Q - \text{diag}(\delta_0 \mathbf{1}_m, \phi(\delta_0) \mathbf{1}_{m'})) w &= w^T \text{diag}(z \mathbf{1}_m, \zeta \mathbf{1}_{m'}) w \\ &+ w^T (Q - \text{diag}((\delta_0 + z) \mathbf{1}_m, \phi(\delta_0 + z) \mathbf{1}_{m'})) w \\ &= w^T \text{diag}((z \mathbf{1}_m, \zeta \mathbf{1}_{m'})) w > 0 \end{aligned}$$

where the strict inequality holds because $\text{diag}(z \mathbf{1}_m, \zeta \mathbf{1}_{m'}) > 0$; hence because $\lambda_1(Q - \text{diag}(\delta_0 \mathbf{1}_m, \phi(\delta_0) \mathbf{1}_{m'})) = 0$, this means that $Q - \text{diag}(\delta_0 \mathbf{1}_m, \phi(\delta_0) \mathbf{1}_{m'})$ must be semidefinite negative and we have a contradiction. \square

Let us denote by Γ the feasibility region of Prob. 5; it is convex [50]. We prove that the search for the optimal solution can be confined to a compact subset of the feasibility region.

Theorem 3 (Compact search set). *There exist two pairs $(\delta_0^{\min}, \delta_0^{\max})$ and $(\delta_1^{\min}, \delta_1^{\max})$ such that a solution $\Delta_2^* = (\delta_0^*, \delta_1^*)$ of Prob. 5 belongs to a compact subset $\Gamma' \subseteq [\delta_0^{\min}, \delta_0^{\max}] \times [\delta_1^{\min}, \delta_1^{\max}]$.*

Proof. Let us define $\hat{c}_0 = \sum_{V_j \in \pi_0} c_0 k_j$ and $\hat{c}_1 = \sum_{V_z \in \pi_1} c_1 k_z$, then we write $U_{l_{\max}} = \hat{c}_0 l_{\max} + \hat{c}_1 l_{\max}$ and $U^* = \hat{c}_0 \delta_0^* + \hat{c}_1 \delta_1^*$.

Let us denote $\Delta_2^{l_{\max}} = (l_{\max}, l_{\max})$, by Thm. 2, $\Delta_2^{l_{\max}} \in \Gamma$, hence $U_{l_{\max}} \geq U^*$ and, by defining set $\Omega = \{(\delta_0, \delta_1) : \hat{c}_0 \delta_0 + \hat{c}_1 \delta_1 \leq U_{l_{\max}}\}$, it follows that $(\delta_0^*, \delta_1^*) \in \Gamma' = \Gamma \cap \Omega$; Γ' is closed as intersection of closed sets.

Now, feasibility conditions of Prob. 5 require matrix $Q - \delta_0 I_m^0 + \delta_1 I_{m'}^1$ to be semidefinite negative. We define $f(\delta_0) = \lambda_1(Q - \delta_0 I_m^0 + (\frac{U_{l_{\max}} - \hat{c}_0 \delta_0}{\hat{c}_1}) I_{m'}^1)$: we have $f(l_{\max}) \leq 0$ since $(l_{\max}, l_{\max}) \in \Gamma$ and $f(0) > 0$ by *i)* of Prop. 3.

By assertion *ii)* in Prop. 3, $f(\delta_0)$ is a continuous function. Hence, there exists δ_0^{\min} such that $f(\delta_0^{\min}) = 0$, and since ϕ is decreasing $\phi(\delta_0^{\min}) = \delta_1^{\max}$. We can repeat the same reasoning by inverting the role of δ_1 and δ_0 defining $g(\delta_1) = \lambda_1(Q - (\frac{U_{l_{\max}} - \hat{c}_1 \delta_1}{\hat{c}_0}) I_m^0 + \delta_1 I_{m'}^1)$. Hence, we can assert that exists δ_1^{\min} such that $g(\delta_1^{\min}) = 0$ and $\phi(\delta_1^{\min}) = \delta_0^{\max}$.

Finally, by letting $r : \hat{c}_0 \delta_0 + \hat{c}_1 \delta_1 = U_{l_{\max}}$, the points $(\delta_0^{\min}, \delta_1^{\max})$ and $(\delta_0^{\max}, \delta_1^{\min})$ belong to $\partial \Gamma \cap r$, i.e., they belong to $\partial \Gamma'$, so $\Gamma' \subseteq [\delta_0^{\min}, \delta_0^{\max}] \times [\delta_1^{\min}, \delta_1^{\max}]$, and consequently, being Γ' closed, it is also compact. \square

Remark 3. *Thm. 3 allows us to identify an interval of the values of δ_0 and δ_1 where we can restrict the search of (δ_0^*, δ_1^*) . Since $\Gamma' \subseteq [\delta_0^{\min}, \delta_0^{\max}] \times [\delta_1^{\min}, \delta_1^{\max}]$ and $(\delta_0^*, \delta_1^*) \in \Gamma'$, then $\delta_0^* \in [\delta_0^{\min}, \delta_0^{\max}]$ and $\delta_1^* \in [\delta_1^{\min}, \delta_1^{\max}]$. This is one key property in the algorithmic search of the optimal solution presented in the following section.*

Finally, a direct proof that the optimal solution lies on $\partial \Gamma'$ follows:

Corollary 1. *A solution $\Delta_2^* = (\delta_0^*, \delta_1^*)$ of Prob. 5 belongs to $\partial \Gamma' \cap \Omega$.*

Proof. Let us assume $\Delta_2^* = (\delta_0^*, \delta_1^*) \in \Gamma' \setminus \partial \Gamma'$. Δ_2^* is feasible, hence $\lambda_1(Q - D) < 0$, with $D = \text{diag}(\delta_0^* \mathbf{1}_m, \delta_1^* \mathbf{1}_{m'})$. From Prop. 3 *ii)*, again we can find $0 < \delta'_1 < \delta_1^*$ such that $\lambda_1(Q - \text{diag}(\delta_0^* \mathbf{1}_m, \delta'_1 \mathbf{1}_{m'})) = 0$, where, i.e., $\Delta'_2 = (\delta_0^*, \delta'_1) \in \partial \Gamma'$. But, $U(\Delta_2^*) - U(\Delta'_2) = \hat{c}_1(\delta_1^* - \delta'_1) > 0$. Contradiction. \square

E. Bisection Algorithm

Tab. I reports on the pseudocode of algorithm `OptimalImmunization2D`: it solves the 2D curing problem. It employs three additional functions `LeftCorner` (Tab. II) `RightCorner` and `BisectionThreshold` (Tab. III).

`LeftCorner` identifies via bisection feasible point $(\delta_0^{\min}, \delta_1^{\max})$; the bisection search operated by `LeftCorner` – see proof of Thm. 3 – is performed along values $\delta_1 = f(\delta_0)$. The companion function `RightCorner` identifies the point $(\delta_0^{\max}, \delta_1^{\min})$. But, the pseudocode is omitted for the sake of space.

TABLE I: OptimalThreshold2D: solves the 2D optimal curing problem via the bisection search.

$(\delta_0^*, \delta_1^*) = \text{OptimalThreshold2D}(Q, c_0, c_1)$	
Receives:	Q, c_0, c_1
Returns:	δ_0^*, δ_1^*
Initialize:	$(\delta_l, \delta_1^{\max}) = \text{LeftCorner}(Q, c_0, c_1)$ $(\delta_1^{\min}, \delta_r) = \text{RightCorner}(Q, c_1, c_0)$ $k \leftarrow 1, U_{k-1} \leftarrow 0, U_k \leftarrow \infty$
1:	WHILE $ U_k - U_{k-1} > \epsilon$
2:	$\delta_0^* = (\delta_l + \delta_r)/2$
3:	$\delta_1^* \leftarrow \text{BisectionThreshold}(Q, \delta_0^*)$
4:	$U_{k+1} = \hat{c}_0 \delta_0^* + \hat{c}_1 \delta_1^*$
5:	IF $\partial U_k < 0$ % (see Rem. 4)
6:	THEN $\delta_r = \delta_0^*$
7:	ELSE $\delta_l = \delta_0^*$
8:	END
9:	$k \leftarrow k + 1$
9:	END

Procedure `isNegativeDefinite` is the standard test for a real symmetric matrix A to be negative definite; it requires to verify $\det(A_k) = (-1)^k$ where A_k is the k -th principal minor of A , i.e., the matrix obtained considering the first k rows and columns only.

Finally, the `OptimalImmunization2D` algorithm performs a bisection search based on a subgradient descent over the utility function $U(\delta_0) = \hat{c}_0 \delta_0 + \hat{c}_1 \phi(\delta_0)$.

Remark 4. In Tab. I we have reported an implementation assuming the calculation of the subgradient ∂U at each mid point x . However, it is sufficient to evaluate the increment at a point $x + \epsilon_1$ within the feasibility region for some $\epsilon_1 > 0$: if $U(x) < U(x + \epsilon_1)$, then, due to convexity, the whole interval $[x + \epsilon_1, +\infty)$ can be discarded. Conversely, if $U(x) > U(x + \epsilon_1)$, then, due to convexity, the whole interval $[0, x)$ can be discarded during the search. This operation can be performed at a cost $O(1)$ when $U(x)$ and $U(x + \epsilon_1)$ are known, i.e., at the cost of two calls of `BisectionThreshold`.

We note that REPEAT loop stops when $\epsilon > \prod |\lambda_i| = |\det(Q - D)| > |\lambda_1|^n$, i.e., when $|\lambda_1| < (\epsilon)^{1/n}$. Furthermore, the termination condition in `BisectionThreshold`, `LeftCorner` and `RightCorner` requires Δ_2 to lie within the feasible region and the determinant to be smaller than ϵ .

Theorem 4 (Correctness). *OptimalThreshold2D is an ϵ approximation of an optimal solution Δ_2^* .*

Proof. The algorithm operates a bisection search for a global minimum of $U(\Delta_2) = \hat{c}_1 \delta_0 + \hat{c}_1 \phi(\delta_0)$, where $U(\Delta_2)$ is a convex function. Let $V = U_{l_{\max}}$: from the properties of the bisection search on (quasi-)convex functions [50][Ch. 4, pp. 145], the accuracy at step $r = \lfloor V/\epsilon \rfloor$ of the algorithm is $U_r - U(\Delta_2^*) < 2^{-r} V = \epsilon$. \square

Furthermore, we can characterize the computational complexity of the algorithm.

Theorem 5 (Complexity). *The time complexity of OptimalThreshold2D is $O(\epsilon^{-2} n^{1+\ell} \log^2 n)$ where $\ell = 2.373$.*

TABLE II: LeftCorner: identifies the left corner of $\Gamma' \subseteq \Gamma$ (Thm. 3); the pseudocode of the dual function $(\delta_0^{\max}, \delta_1^{\min}) = \text{RightCorner}(Q, c_0, c_1)$ is omitted for the sake of space.

$(\delta_0^{\min}, \delta_1^{\max}) = \text{LeftCorner}(Q, c_0, c_1)$	
Receives:	Q, c_0, c_1
Returns:	δ_0^{\min}
Initialize:	$U_{\max} \leftarrow (\hat{c}_0 + \hat{c}_1) l_{\max}$
1:	REPEAT
2:	$\delta_0^{\min} = (\delta_l + \delta_r)/2$
3:	$\delta_1^{\max} \leftarrow \frac{U_{\max} - \hat{c}_0 \delta_0^{\min}}{c_1}$
4:	$D = \text{diag}(\delta_0^{\min} \mathbf{b} f \mathbf{1}_m, \delta_1^{\max} \mathbf{1}_{m'})$
5:	$X \leftarrow \text{isNegativeDefinite}(Q - D)$
6:	IF $X = \text{true}$
7:	THEN $\delta_r = \delta_0^*$ % discard larger values
8:	ELSE $\delta_l = \delta_0^*$ % discard smaller values
9:	END
10:	$T = \det(Q - D)$
12:	UNTIL $X == \text{TRUE}$ AND $ T < \epsilon$ % Termination condition

TABLE III: BisectionThreshold: given feasible δ_0 , finds δ_1 such that (δ_0, δ_1) lies on the frontier of the feasibility region.

$\delta_1 = \text{BisectionThreshold}(Q, \delta_0)$	
Receives:	Q, δ_0
Returns:	δ_1
Initialize:	$T \leftarrow \inf, \delta_l = 0, \delta_r \leftarrow \max_i \sum_j a_{ij}$
1:	REPEAT
2:	$\delta_1 = (\delta_l + \delta_r)/2$
3:	$D \leftarrow \text{diag}(\delta_0 \mathbf{1}_m, \delta_1 \mathbf{1}_{m'})$
4:	$X \leftarrow \text{isNegativeDefinite}(Q - D)$
5:	IF $X = \text{true}$
6:	THEN $\delta_r = \delta_1$ % discard larger values
7:	ELSE $\delta_l = \delta_1$ % discard smaller values
8:	END
9:	$T = \det(Q - D)$
10:	UNTIL $X == \text{TRUE}$ AND $ T < \epsilon$ % Termination condition

Proof. The number of iterations of the bisection search WHILE loop (lines 1 to 9 in Tab. I) is $O(\epsilon^{-1} \log n)$. This follows again from elementary properties of bisection search [50][Ch. 4, pp. 145]. In fact, the bisection search operates for $0 \leq U(\delta_0) \leq U_{l_{\max}}$ and $U_{l_{\max}} = l_{\max}(\hat{c}_0 + \hat{c}_1)$. Finally, indeed, $l_{\max} \leq (n-1) \max_{i,j} q_{ij}$.

Using the same argument on the measure of the search intervals of `BisectionThreshold`, `LeftCorner` and `RightCorner` we conclude that they require $O(\epsilon^{-1} \log n)$ iterations of the REPEAT loop as well.

Finally, test `isNegativeDefinite` appearing in `Threshold2D`, `LeftCorner` and `RightCorner` requires the computation of $n-1$ determinants of the principal minors of $A - D$ at cost $O(n^{1+\ell})$. Here ℓ is the exponent for fast matrix multiplication [57]. In the case of the Coppersmith-Winograd algorithm for fast matrix multiplication it holds $\ell = 2.373$. \square

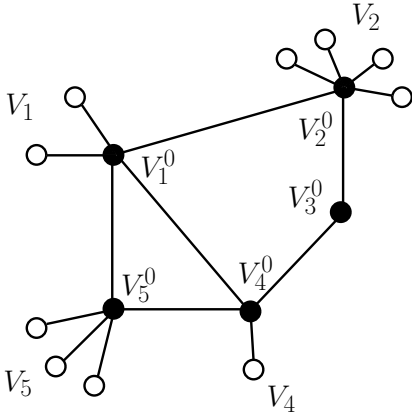


Fig. 1: A sample inteconnected star network with $n = 10$ communities, and $k_1 = 2, k_2 = 4, k_3 = 0, k_4 = 1, k_5 = 3$; central communities are filled black, terminal communities are filled white.

F. Numerical Results

In this section we present the result of numerical experiments in the case of interconnected stars networks, a sample network is depicted in Fig. 1.

In Figure 2a) we compare the ratio between the cost U_u of the uniform curing rate vector, and the optimal cost $U^* = U(\Delta^*)$ solving the 2D curing Prob. 5. The uniform curing rate vector is $\Delta = \delta \mathbf{1}_N$ where δ is the value such that the threshold in (13) is attained. For this experiment we consider that the infection spreads with rate $\beta_{V_i^0 V_j^0} = \beta_0$ among the central communities and with rate $\beta_{V_i^0 V_i} = \beta_1$ between a central node and a node in its adjacent terminal community, moreover we assume $c_0 = c_1 = 1$.

We consider that each terminal community has the same number of elements k . The computation is made for different values of k , for three different sample networks, with $m = 50$ central nodes and $m' = 50$ terminal communities. Sample networks differ for the average degree of the central nodes. Central nodes are connected as Erdős-Rényi sample graphs with $p = 0.2, p = 0.3, p = 0.6$, respectively.

The plot confirms that a larger gain is obtained by 2D curing policy versus a uniform approach, in particular, the larger the denser the network, namely, for larger p in our samples. For the interconnected stars networks samples, in particular, we observe one order of magnitude gain in the cost function. We see that the advantage increases as the number of elements k increases, with a \sqrt{k} shaped ratio (20) as derived in closed form for the case $m = m' = 2$. In Figure 2b) we have instead reported, only, on the optimal cost U^* for different values of c_0 and c_1 . In particular, we observe that the optimal cost appears to depend linearly on the community size k .

Larger costs are incurred in the case when the curing cost of a central node is larger than the curing cost of a terminal one. This is in line with the fact that central communities are more connected than terminal communities, and consequently we need for more investments in such a way that the infection is kept subcritical.

In Tab. IV we compare the performance of `OptimalThreshold2D` with a SDP solver, namely the SDPT3 solver [53]. The SDPT3 solver generates a solution using a primal-dual interior-point algorithm which leverages on infeasible path-following paradigm.

As reported in Tab. IV), when the solver is applied to Prob. 5, we denote the corresponding solution as SDPT3 (2D). For the sake of comparison we have reported also on the optimal solution derived with the same solver when curing rates are optimized per node (Prob. 2), and we refer to this solution as SDPT3. The solution is provided on a graph with $m = 50$ central nodes and for $c_0 = c_1 = 1$, for increasing values of the terminal community dimension k .

We can observe that for the interconnected stars network, in the case of two infection rate levels, SDPT3, SDPT3 (2D) and `OptimalThreshold2D` provide similar values. This result suggests that, in this particular case, there is no advantage to treat each node with different curing policies: the general solution obtained using SDPT3 has same performance as the one generated solving the 2D formulation of the problem, i.e., by using `OptimalThreshold2D` or SDPT3 in the 2D case. We observe also that the immunization rate of terminal nodes appears insensitive to the increase of the terminal communities size k , as it can be seen, with a direct computation, in example IV-C for the case with $m = 1$ and $m = 2$.

In Tab. V, same performance evaluation has been reported for the same sample graph as above, but studying the case with more than two infection rate levels; specifically a central node can eventually infect each of its adjacent central node with a different infection rate, also the infection rate between a central node and a terminal community can vary from a subgraph to another. The infection rates are generated as uniform random variables with mean $\beta_0 = 1$ between the hubs and $\beta_1 = 0.3$ for each subgraph (the results are averaged over 10 instances). As seen, by curing nodes with different curing policies it is possible to attain lower costs at larger values of k . This effect is depicted also in Fig. 3. In particular, again, the 2D curing rates output of SDPT3 (2D) and `OptimalThreshold2D` show similar performance and the optimal curing rate of terminal communities appears insensitive to the increase of the terminal communities size.

G. Conclusions

We use the SIS model to study the diffusion of epidemics over heterogeneous networks. Such networks are directed weighted graphs capturing the interaction between individuals in a population and the large heterogeneity in the intensity of the connections.

Using a meanfield approximation, it is possible to provide necessary and sufficient conditions for the extinction of the epidemics by studying the sign of the stability modulus of the graph. Then, we have specialized the theory to the case of equitable partitions in order to model heterogeneous community networks.

Finally, it is possible to leverage on the previous framework in order to provide an optimal curing policy, for preventing the

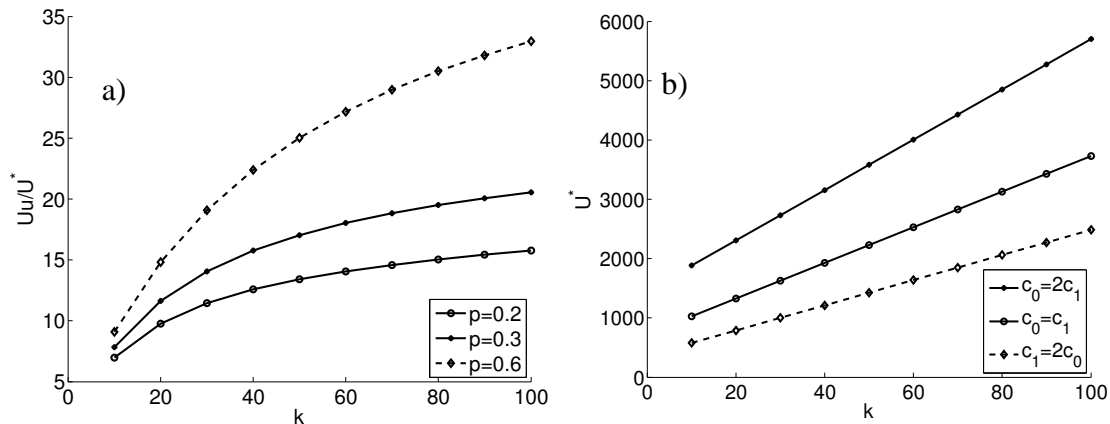


Fig. 2: (a) Ratio U_u/U^* for increasing size k of the terminal communities: $\beta_0 = 1$, $\beta_1 = 0.3$, $c_0 = c_1 = 1$. The three curves refer to interconnected star networks with $m = m' = 50$, where the central nodes are connected as Erdős-Rényi network generated for $p = 0.2$, $p = 0.3$ and $p = 0.6$ respectively. (b) Cost function U^* for increasing dimension k of the terminal communities, $\beta_0 = 1$, $\beta_1 = 0.3$. The curves refer to the case $p = 0.3$ in the cases $c_0 = 2c_1$, $c_0 = c_1$ and $2c_0 = c_1$, respectively.

TABLE IV: Performance of OptimalThreshold2D, SDPT3 (2D) and SDPT3 for a Erdős-Rényi graph generated with $p = 0.2$ with $c_0 = c_1 = 1$; the weights $\beta_0 = 1$, and $\beta_1 = 0.3$.

k	OptimalThreshold2D			SDPT3 (2D)			SDPT3		
	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$
10	0.8057	13.116	0.29979	0.80572	13.1144	0.3	0.772	12.44	0.3
20	1.1057	16.1163	0.29984	1.1057	16.1144	0.3	1.072	15.44	0.3
30	1.4056	19.1222	0.29965	1.4057	19.1145	0.3	1.372	18.44	0.3
40	1.7055	22.1294	0.29951	1.7057	22.1144	0.3	1.672	21.44	0.3
50	2.0053	25.1354	0.29943	2.0057	25.1144	0.3	1.972	24.44	0.3
60	2.3052	28.1414	0.29936	2.3057	28.1144	0.3	2.272	27.44	0.3
70	2.6049	31.1578	0.29916	2.6057	31.1145	0.3	2.572	30.44	0.3
80	2.9047	34.1792	0.29893	2.9057	34.1144	0.3	2.872	33.4401	0.3
90	3.2043	37.1929	0.29882	3.2057	37.1144	0.3	3.172	36.4402	0.3
100	3.5039	40.1319	0.29947	3.5057	40.1144	0.3	3.472	39.44	0.3

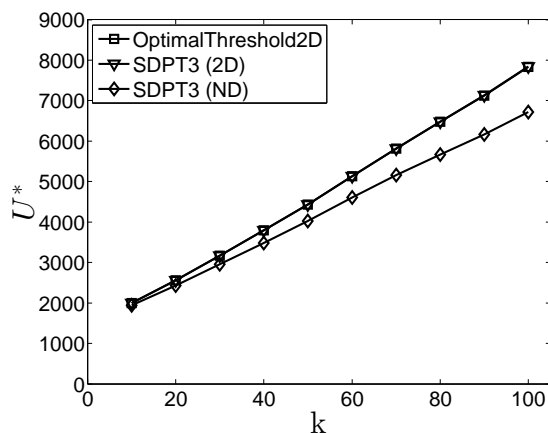


Fig. 3: Detail of the cost in the case of uniform distribution of infection rates.

epidemics to persist indefinitely in the population, taking into account the structure of the network. In particular we focused on the problem of distributing different amount of resources in different communities (hospitals, villages, cities,...), that is an adequate way to represent policy-makers decisions, rather

than to consider policies for each individual agent.

At last we have discussed on the special case of a two-dimensional curing policy, that can reflect, e.g., the case of different policy decisions for two different kind of individuals (male and female, younger and elders, etc.,...). With respect to this problem we have presented an ϵ -approximation algorithm with polynomial complexity in the input size.

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APPENDIX

Proposition 2. Let A be an $n \times n$ irreducible and non-negative matrix and let $D = \text{diag}(\delta_1, \dots, \delta_n)$. Then it holds:

- $A - D$ is irreducible, for each $(\delta_1, \dots, \delta_n)$.
- There exists an eigenvector w of $A - D$ such that $w > 0$ (i.e. each component $w_i > 0$, $i = 1, \dots, n$) and the corresponding eigenvalue is $r(A - D)$, for each $(\delta_1, \dots, \delta_n)$.

TABLE V: Performance of OptimalThreshold2D, sample graphs are obtained from the same graph used in Tab. IV and infection rates are generated as uniform random variables with mean $\beta_0 = 1$ and $\beta_1 = 0.3$, for the speed of infection between central communities and between a central node and a terminal community respectively.

k	OptimalThreshold2D			SDPT3 (2D)			SDPT3		
	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$	$U^*(10^3)$	δ_0^*	$k \cdot \delta_1^*$
10	1.9963	34.1309	0.57945	1.9963	34.1309	0.57945	1.9379	33.4102	0.53481
20	2.5603	39.3993	0.5903	2.5603	39.3993	0.5903	2.4307	38.3384	0.51382
30	3.1665	44.9001	0.61431	3.1666	44.9001	0.61431	2.9536	43.5669	0.51682
40	3.7954	50.6431	0.63164	3.7957	50.6431	0.63164	3.4781	48.8125	0.51876
50	4.4332	56.4137	0.64499	4.4336	56.4137	0.64499	4.0279	54.3098	0.52495
60	5.1272	62.627	0.66528	5.1278	62.627	0.66528	4.6049	60.0804	0.53364
70	5.8175	69.0209	0.67612	5.8184	69.0209	0.67612	5.1595	65.6263	0.53663
80	6.4779	74.8633	0.68369	6.4792	74.8633	0.68369	5.6708	70.7388	0.53346
90	7.1277	80.4688	0.68984	7.1298	80.4688	0.68984	6.1686	75.717	0.5295
100	7.8361	87.1312	0.6959	7.839	87.1312	0.6959	6.7182	81.2132	0.53151

Proof. i. From [22]: a $n \times n$ matrix A is said to be irreducible if for any proper subset $S \subseteq \{1, \dots, n\}$ there exists $i \in S$ and $j \in S' = \{1, \dots, n\} - S$ such that $a_{ij} \neq 0$; since A is irreducible, the definition applies immediately to $A - D$;
ii. See [22, Lemma 4.2]. \square

Proposition 3. *Let A be an $n \times n$ symmetric, irreducible and non negative matrix and let $D = \text{diag}(\delta_1, \dots, \delta_n)$. Then it holds:*

- i. *Let $\delta_i = 0$ for some $i = 1, \dots, n$, then $\lambda_1(A - D) \geq 0$.*
- ii. *The function $(\delta_1, \dots, \delta_n) \mapsto \lambda_1(D - A)$ is continuous.*

Proof. i. Let consider $\delta_i = 0$ for some $i = 1, \dots, n$ and assume by contradiction that $\lambda_1(A - D) < 0$, this means that the matrix $A - D$ must be definite negative; however if we take the vector \mathbf{e}_i of the canonical basis of \mathbb{R}^n , then it holds that $\mathbf{e}_i^T(A - D)\mathbf{e}_i = \mathbf{e}_i^T A \mathbf{e}_i \geq 0$ and we have a contradiction.
ii. It follows since the eigenvalues of a matrix A vary with continuity with the entries of A , see [58, Appendix D]. \square

REFERENCES

- [1] P. Van Mieghem and J. Omic, "In-homogeneous virus spread in networks," *arxiv:1306.2588*, 2013.
- [2] B. Qu and H. Wang, "Sis epidemic spreading with heterogeneous infection rates," *arXiv preprint arXiv:1506.07293*, 2015.
- [3] A. Widder and C. Kuehn, "Heterogeneous population dynamics and scaling laws near epidemic outbreaks," *arXiv preprint arXiv:1411.7323*, 2014.
- [4] M. E. Newman, "The structure and function of complex networks," *SIAM review*, vol. 45, no. 2, pp. 167–256, 2003.
- [5] D. Mugnolo, *Semigroup methods for evolution equations on networks*. Springer, 2014.
- [6] V. M. Preciado, M. Zargham, C. Enyioha, A. Jadbabaie, and G. Pappas, "Optimal vaccine allocation to control epidemic outbreaks in arbitrary networks," *CoRR*, vol. abs/1303.3984, 2013.
- [7] P. Van Mieghem, J. Omic, and R. Kooij, "Virus spread in networks," *Networking, IEEE/ACM Tran. on*, vol. 17, pp. 1–14, Feb 2009.
- [8] P. L. Simon, M. Taylor, and I. Z. Kiss, "Exact epidemic models on graphs using graph-automorphism driven lumping," *Journal of mathematical biology*, vol. 62, no. 4, pp. 479–508, 2011.
- [9] F. D. Sahneh, C. Scoglio, and P. Van Mieghem, "Generalized epidemic mean-field model for spreading processes over multi-layer complex networks," *Networking, IEEE/ACM Transactions on*, vol. 21, no. 5, pp. 1609–1620, 2013.
- [10] P. Van Mieghem, "The n-intertwined sis epidemic network model," *Computing*, vol. 93, no. 2-4, pp. 147–169, 2011.
- [11] I. Nåsell, "The quasi-stationary distribution of the closed endemic sis model," *Advances in Applied Probability*, pp. 895–932, 1996.
- [12] I. Nåsell, "Stochastic models of some endemic infections," *Mathematical biosciences*, vol. 179, no. 1, pp. 1–19, 2002.
- [13] A. Ganesh, L. Massoulié, and D. Towsley, "The effect of network topology on the spread of epidemics," in *INFOCOM 2005. 24th Annual Joint Conference of the IEEE Computer and Communications Societies. Proceedings IEEE*, vol. 2, pp. 1455–1466, IEEE, 2005.
- [14] R. van de Bovenkamp and P. Van Mieghem, "Survival time of the susceptible-infected-susceptible infection process on a graph," *Physical Review E*, vol. 92, no. 3, p. 032806, 2015.
- [15] P. Van Mieghem, "Decay towards the overall-healthy state in sis epidemics on networks," *arXiv preprint arXiv:1310.3980*, 2013.
- [16] M. Draief and L. Massouli, *Epidemics and rumours in complex networks*. Cambridge University Press, 2010.
- [17] P. Van Mieghem, "Exact markovian sir and sis epidemics on networks and an upper bound for the epidemic threshold," *arXiv preprint arXiv:1402.1731*, 2014.
- [18] P. Van Mieghem, "Exact markovian sir and sis epidemics on networks and an upper bound for the epidemic threshold," *arXiv preprint arXiv:1402.1731*, 2014.
- [19] E. Cator and P. Van Mieghem, "Nodal infection in Markovian SIS and SIR epidemics on networks are non-negatively correlated," *Physical Review E*, vol. 89, no. 5, p. 052802, 2014.
- [20] P. Van Mieghem and R. Van de Bovenkamp, "Accuracy criterion for the mean-field approximation in susceptible-infected-susceptible epidemics on networks," *Physical Review E*, vol. 91, no. 3, p. 032812, 2015.
- [21] P. Van Mieghem, "Approximate formula and bounds for the time-varying susceptible-infected-susceptible prevalence in networks," *Physical Review E*, vol. 93, no. 5, p. 052312, 2016.
- [22] A. Lajmanovich and J. A. Yorke, "A deterministic model for Gonorrhea in a non-homogeneous population," *Mathematical Biosciences*, vol. 28, no. 34, pp. 221 – 236, 1976.
- [23] S. Bonaccorsi, S. Ottaviano, D. Mugnolo, and F. De Pellegrini, "Epidemic outbreaks in networks with equitable or almost-equitable partitions," *SIAM Journal of Applied Mathematics*, vol. 75, no. 6, pp. 2421 – 2443, 2015.
- [24] S. Boccaletti, V. Latora, Y. Moreno, M. Chavez, and D.-U. Hwang, "Complex networks: Structure and dynamics," *Physics reports*, vol. 424, no. 4, pp. 175–308, 2006.
- [25] I. Hanski and O. Ovaskainen, "Metapopulation theory for frag-

- mented landscapes,” *Theoretical population biology*, vol. 64, no. 1, pp. 119–127, 2003.
- [26] N. Masuda, “Effects of diffusion rates on epidemic spreads in metapopulation networks,” *New Journal of Physics*, vol. 12, no. 9, p. 093009, 2010.
- [27] L. Allen, B. Bolker, Y. Lou, and A. Nevai, “Asymptotic profiles of the steady states for an sis epidemic patch model,” *SIAM Journal on Applied Mathematics*, vol. 67, no. 5, pp. 1283–1309, 2007.
- [28] F. Ball, D. Mollison, and G. Scalia-Tomba, “Epidemics with two levels of mixing,” *The Annals of Applied Probability*, pp. 46–89, 1997.
- [29] J. V. Ross, T. House, and M. J. Keeling, “Calculation of disease dynamics in a population of households,” *PLoS One*, vol. 5, no. 3, p. e9666, 2010.
- [30] F. Ball and P. Neal, “A general model for stochastic sir epidemics with two levels of mixing,” *Mathematical biosciences*, vol. 180, no. 1, pp. 73–102, 2002.
- [31] F. Ball, T. Britton, T. House, V. Isham, D. Mollison, L. Pellis, and G. S. Tomba, “Seven challenges for metapopulation models of epidemics, including households models,” *Epidemics*, vol. 10, pp. 63–67, 2015.
- [32] L. Pellis, F. Ball, and P. Trapman, “Reproduction numbers for epidemic models with households and other social structures. i. definition and calculation of r_0 ,” *Mathematical biosciences*, vol. 235, no. 1, pp. 85–97, 2012.
- [33] F. Ball and P. Neal, “Network epidemic models with two levels of mixing,” *Mathematical biosciences*, vol. 212, no. 1, pp. 69–87, 2008.
- [34] B. Frank, S. David, T. Pieter, *et al.*, “Threshold behaviour and final outcome of an epidemic on a random network with household structure,” *Advances in Applied Probability*, vol. 41, no. 3, pp. 765–796, 2009.
- [35] F. Ball, D. Sirl, and P. Trapman, “Analysis of a stochastic sir epidemic on a random network incorporating household structure,” *Mathematical Biosciences*, vol. 224, no. 2, pp. 53–73, 2010.
- [36] H. Wang, Q. Li, G. D’Agostino, S. Havlin, H. E. Stanley, and P. Van Mieghem, “Effect of the interconnected network structure on the epidemic threshold,” *Physical Review E*, vol. 88, no. 2, p. 022801, 2013.
- [37] P. Van Mieghem, “Interconnectivity structure of a general interdependent network,” *Physical Review E*, vol. 93, no. 4, p. 042305, 2016.
- [38] S. Bonaccorsi, S. Ottaviano, F. De Pellegrini, A. Socievole, and P. Van Mieghem, “Epidemic outbreaks in two-scale community networks,” *Phys. Rev. E*, vol. 90, p. 012810, Jul 2014.
- [39] A. J. Schwenk, “Computing the characteristic polynomial of a graph,” in *Graphs and Combinatorics*, pp. 153–172, Springer, 1974.
- [40] C. Godsil, “Feasibility conditions for the existence of walk-regular graphs,” *Linear Algebra and its Applications*, vol. 30, pp. 15–61, 1980.
- [41] B. A. Prakash, L. Adamic, T. Iwashyna, H. Tong, and C. Faloutsos, “Fractional immunization in networks,” *Austin, Texas, USA*, 2013.
- [42] C. Borgs, J. Chayes, A. Ganesh, and A. Saberi, “How to distribute antidote to control epidemics,” *Random Structures & Algorithms*, vol. 37, no. 2, pp. 204–222, 2010.
- [43] Y. Wan, S. Roy, and A. Saberi, “Designing spatially heterogeneous strategies for control of virus spread,” *Systems Biology, IET*, vol. 2, no. 4, pp. 184–201, 2008.
- [44] E. Gourdin, J. Omic, and P. Van Mieghem, “Optimization of network protection against virus spread,” in *Design of Reliable Communication Networks (DRCN), 2011 8th International Workshop on the*, pp. 86–93, IEEE, 2011.
- [45] F. D. Sahneh and C. M. Scoglio, “Optimal information dissemination in epidemic networks,” in *Decision and Control (CDC), 2012 IEEE 51st Annual Conference on*, pp. 1657–1662, IEEE, 2012.
- [46] K. Drakopoulos, A. Ozdaglar, and J. N. Tsitsiklis, “When is a network epidemic hard to eliminate?,” *arXiv preprint arXiv:1510.06054*, 2015.
- [47] K. Drakopoulos, A. Ozdaglar, and J. N. Tsitsiklis, “A lower bound on the performance of dynamic curing policies for epidemics on graphs,” *arXiv preprint arXiv:1510.06055*, 2015.
- [48] N. Hupert, J. Cuomo, M. A. Callahan, A. I. Mushlin, and S. S. Morse, *Community-based mass prophylaxis: A planning guide for public health preparedness*. US Department of Health and Human Services, Agency for Healthcare Research and Quality, 2004.
- [49] S. P. Boyd, “Semidefinite programming,” *SIAM review*, vol. 38, pp. 49–95, 1994.
- [50] S. Boyd and L. Vandenberghe, *Convex optimization*. Cambridge University Press, 2004.
- [51] V. M. Preciado, M. Zargham, C. Enyioha, A. Jadbabaie, and G. J. Pappas, “Optimal resource allocation for network protection against spreading processes,” *Control of Network Systems, IEEE Transactions on*, vol. 1, no. 1, pp. 99–108, 2014.
- [52] C. Enyioha, A. Jadbabaie, V. Preciado, and G. J. Pappas, “Distributed resource allocation for epidemic control,” *arXiv preprint arXiv:1501.01701*, 2015.
- [53] R. H. Tütüncü, K. C. Toh, and M. J. Todd, “Solving semidefinite-quadratic-linear programs using SDPT3,” *Mathematical Programming*, vol. 95, no. 2, pp. 189–217, 2003.
- [54] A.-L. Barabási and R. Albert, “Emergence of scaling in random networks,” *science*, vol. 286, no. 5439, pp. 509–512, 1999.
- [55] C. Li, R. van de Bovenkamp, and P. Van Mieghem, “Susceptible-infected-susceptible model: A comparison of n-intertwined and heterogeneous mean-field approximations,” *Physical Review E*, vol. 86, no. 2, p. 026116, 2012.
- [56] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, “Epidemic processes in complex networks,” *Reviews of modern physics*, vol. 87, no. 3, p. 925, 2015.
- [57] A. Aho, J. Hopcroft, and J. Ullman, *The Design and Analysis of Computer Algorithms*. Addison-Wesley, 1974.
- [58] R. A. Horn and C. R. Johnson, eds., *Matrix Analysis*. New York, NY, USA: Cambridge University Press, 2012.

